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## Mononeuritis Multiplex

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### [Author and Editor Disclosure](#)

**Synonyms and related keywords:** mononeuropathy multiplex, multifocal neuropathy, multiple mononeuropathy, peripheral polyneuritis, peripheral mononeuropathy

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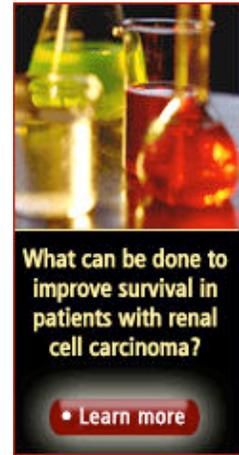
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**Background**

Mononeuritis multiplex is a painful asymmetric asynchronous sensory and motor peripheral neuropathy involving isolated damage to at least 2 separate nerve areas. Multiple nerves in random areas of the body can be affected. As the condition worsens, it becomes less multifocal and more symmetric. Mononeuropathy multiplex syndromes can be distributed bilaterally, distally, and proximally throughout the body.

Mononeuritis multiplex actually is a group of disorders, not a true distinct disease entity. Typically, the condition is associated with (but not limited to) systemic disorders such as diabetes, vasculitis, amyloidosis, direct tumor involvement, polyarteritis nodosa, rheumatoid arthritis, systemic lupus erythematosus, and paraneoplastic syndromes. Mononeuritis multiplex also may be associated with Lyme disease, Wegener's granulomatosis, Sjögren syndrome, cryoglobulinemia, hypereosinophilia, temporal arteritis, scleroderma, sarcoidosis, leprosy, acute viral hepatitis A, and acquired immunodeficiency syndrome (AIDS).

**Pathophysiology**

Mononeuritis multiplex involves damage to at least 2 separate nerve areas. This condition can become progressively worse over time. The damage to the nerves involves destruction of the axon (ie, the part of the nerve cell that is analogous to the copper part of a wire), thus interfering with nerve conduction. Common causes of damage include a lack of oxygen from decreased blood flow or inflammation of blood vessels. Approximately 33% of cases originate from unidentifiable causes.

**Frequency****United States**

The actual incidence in the United States is not known due to the widely varied underlying pathologies that may lead to the disorder. The primary disease process often is so dominant that the symptoms of mononeuritis multiplex simply are attributed to the initial disease and remain undiagnosed.

### International

Same as frequency in the United States (see above).

### Mortality/Morbidity

If the cause is identified early and is successfully treated, full recovery is possible. The extent of disability varies, from no disability to partial or complete loss of function and movement.

### Race

No specific relation to race is known.

### Sex

Mononeuritis multiplex exhibits equal incidence in men and women.

### Age

Age of onset depends on the patient's age at occurrence of the associated disease process; however, this condition does tend to occur in older patients with relatively mild or even unrecognized diabetes for unknown reasons.

## CLINICAL

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### History

A detailed and complete medical history is vitally important in determining the possible underlying cause of the disorder. Pain often begins in the low back or hip and spreads to the thigh and knee on one side. The pain usually is characterized as deep and aching with superimposed lancinating jabs that are most severe at night. Individuals with diabetes typically present with acute onset of unilateral severe thigh pain that is followed rapidly by weakness and atrophy of the anterior thigh muscles and loss of the knee reflex. Other possible symptoms that may be reported by the patient include the following:

- Numbness
- Tingling
- Abnormal sensation
- Burning pain - Dysesthesia
- Difficulty moving a body part - Paralysis
- Lack of controlled movement of a body part

### Physical

Loss of sensation and movement may be associated with dysfunction of specific nerves. Examination reveals preservation

of reflexes and good strength except in regions more profoundly affected. Some common findings of mononeuritis multiplex may include the following (not listed in order of frequency):

- Sciatic nerve dysfunction
- Femoral nerve dysfunction
- Common peroneal nerve dysfunction
- Axillary nerve dysfunction
- Radial nerve dysfunction
- Median nerve dysfunction
- Ulnar nerve dysfunction
- Tardive ulnar palsy
- Peroneal nerve palsy
- Autonomic dysfunction - The part of the nervous system that controls involuntary bodily functions, such as the glands and the heart

## Causes

Mononeuritis multiplex most commonly is associated with diabetes mellitus and multiple nerve compressions.

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## Other Problems to be Considered

Lyme disease  
Sjögren syndrome  
Cryoglobulinemia  
Temporal arteritis  
Scleroderma  
Sarcoidosis  
Leprosy  
Acute viral hepatitis A  
AIDS  
Hypereosinophilia  
Cryoglobulinemia  
Paraneoplastic peripheral neuropathy

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## Lab Studies

- Laboratory tests are ordered, as appropriate, to identify underlying potential causes of the condition. Selection of appropriate tests is guided by the patient's history, physical examination, and symptoms. Examples include the following:
  - If Lyme disease is suspected, draw a *Borrelia burgdorferi* antibody titer.
  - If the human immunovirus is suspected, perform HIV blood tests.
  - If hepatitis is suspected as a causative agent, perform a hepatitis screen.
  - If a systemic inflammatory process is suspected, perform an erythrocyte sedimentation rate (ESR).
- The practitioner must carefully choose screening tests to maximize the likelihood of discovering the causative agent. Choices of screening laboratory tests include, but are not limited to, the following:
  - Complete blood count with a differential
  - Hepatitis screen
  - Blood sugar levels
  - Erythrocyte sedimentation rate

## Imaging Studies

- Imaging studies are not indicated for the diagnosis of mononeuritis multiplex.

## Other Tests

- The suspected cause as suggested by the history, symptoms, and pattern of symptom development helps determine which tests to perform.

- Electrodiagnostic studies are used only in conjunction with an accurate and complete history and physical examination.
  - Sensory nerve conduction studies
    - The lesion or lesions are distal to both the motor and sensory cell bodies and result in either axonal disruption/degeneration or abnormal axonal conduction.
    - Sensory nerve conduction studies (NCS) show abnormalities of decreased amplitude in the presence of axonal disruption. Physical examination will direct the electromyographic examination.
    - Sensory NCS are beyond the reference range for amplitude and/or latency only if a large enough percentage of the sensory axons are damaged. A lesion that eliminates conduction in less than 10% of the sensory axons produces a loss of amplitude that may not be detectable.
    - H-reflex latencies may be prolonged or absent.
  - Motor nerve conduction studies
    - Abnormalities are similar to those seen in axonal polyneuropathies and entrapment neuropathies with the exception of the anatomic distribution. A reduction in the sensory and motor action potential amplitudes and minimal alterations in nerve conduction velocity will be seen. Velocity may be slightly reduced compared with the reference range, but it does not usually decrease below 70-80% of the lower limit of the reference range. Additionally, the loss of motor axons should generate a reduced number of motor unit action potentials (MUAP) firing at high rates (ie, reduced recruitment). Abnormal findings directly depend on the severity and aggressiveness of the underlying disease.
    - A decrement of motor amplitude may be seen if there is significant denervation.
  - Needle electrode examination
    - Results of needle electrode examination can vary, depending on the time course of the disorder.
    - Findings are typically neuropathic and may include abnormal spontaneous membrane activity (positive sharp waves and fibrillation potentials) and increases in MUAP duration, amplitude, and polyphasia.

## Procedures

- The performance of any procedure, such as a nerve biopsy, is dictated by the history and physical examination. The purpose of any procedure is to determine the primary pathologic process.
- A nerve biopsy may be performed to help distinguish between demyelination (destruction of parts of the myelin sheath covering the nerve) and axonal degeneration (destruction of the axon portion of the nerve cell), to identify inflammatory nerve conditions (neuropathies), or to confirm specific diagnoses.

## Histologic Findings

In some cases, a nerve biopsy may be appropriate to determine the underlying cause (eg, a combination of perivascular inflammation and axonal loss or demyelination and axonal loss with multinucleated inflammatory cells). A pattern of necrotizing vasculitis of epineural arteries may be observed in HIV-related mononeuritis.

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## Rehabilitation Program

### Physical Therapy

Physical therapy (PT) may be recommended for patients with mononeuritis multiplex, and the specific treatment generally depends on the site involved. PT can help to prevent contractures and maintain strength by instructing the patient in range of motion (ROM) and strengthening exercises as appropriate. The physical therapist can assist the patient with positioning issues and recommend braces or splints (static and functional) to increase his/her independence with mobility.

Patients with mononeuritis multiplex often exhibit problems with diminished sensation and require instruction to improve their safety awareness. In some cases, a transcutaneous electrical nerve stimulation (TENS) unit may be recommended for pain control in patients with this condition. The physical therapist can instruct the patient in the appropriate setup and use of the unit.

### Occupational Therapy

Occupational therapy is directed toward maintaining functional independence in patients with mononeuritis multiplex. This training may include instructing the patient in the use of adaptive techniques for activities of daily living (ADL). Built-up handles on eating utensils and adaptive aids (eg, long shoehorns, reachers) may be used to help the patient perform ADL such as dressing and eating. A job-site analysis may need to be completed by the physical therapist and/or occupational therapist to ensure occupational safety. Therapy can incorporate job-specific training for those individuals who would benefit.

### Speech Therapy

Speech therapy usually is not required for patients with mononeuritis multiplex.

### Recreational Therapy

The primary focus of recreational therapy is to maintain the patient's activity level and self-esteem during recovery.

## Medical Issues/Complications

The physician must try to elucidate the underlying cause and initiate appropriate treatment.

- Disorders such as vasculitis can be fatal if not treated.
- Use caution in treating the patients who are insensate, especially with the use of modalities (eg, ice, heat).
- Monitor and help control blood sugar levels in individuals with diabetes.
- Institute nutritional supplementation.
- Monitor bony prominences for pressure points.
- Safety is an important consideration, and appropriate safety measures must be provided. Safety measures may include installation of railings, removal of obstacles (eg, loose rugs that may slip on the floor), installation of low-level lighting, testing of water temperatures before bathing, and implementation of other measures, as appropriate.

## Consultations

- Neurologist - If an underlying neurologic condition is suspected
- Rheumatologist - If an underlying rheumatologic condition is suspected

- Infectious disease specialist- If evidence of an infectious etiology is present

## Other Treatment

Most treatments are directed toward management of the underlying condition. Treatments should be directed according to the established protocols for those specific disease conditions.

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The reduction of inflammation around the epineurium is the primary goal of treatment. Additional treatments are aimed at treating the primary cause and/or associated symptomology.

### Drug Category: *Corticosteroids*

Can be helpful if the condition is due to vasculitis. Failure to recognize and treat vasculitis can result in fatal consequences.

<b>Drug Name</b>	Prednisone (Deltasone, Orasone, Meticorten)
<b>Description</b>	Immunosuppressant for treatment of autoimmune disorders; may decrease inflammation by reversing increased capillary permeability and suppressing PMN activity. Stabilizes lysosomal membranes and also suppresses lymphocytes and antibody production. Long-term treatment with corticosteroids may be necessary in individuals with mononeuritis multiplex.
<b>Adult Dose</b>	5-60 mg/d PO qd or divided bid/qid; taper over 2 wk, as symptoms resolve
<b>Pediatric Dose</b>	4-5 mg/m <sup>2</sup> /d PO; alternatively, 0.05-2 mg/kg PO divided bid/qid; taper over 2 wk, as symptoms resolve
<b>Contraindications</b>	Documented hypersensitivity; systemic viral, fungal, or tubercular infections
<b>Interactions</b>	Prednisone clearance may decrease when used concurrently with estrogens; use with digoxin may increase digitalis toxicity secondary to hypokalemia; phenobarbital, phenytoin, and rifampin also may increase the metabolism of glucocorticoids; consider increasing the maintenance dose; monitor patients for

	hypokalemia when taking this medication concurrently with diuretics
<b>Pregnancy</b>	C - Safety for use during pregnancy has not been established.
<b>Precautions</b>	Long-term use of corticosteroids may predispose patients to various problems including hyperglycemia, manifestation of latent diabetes mellitus, nonketotic hyperosmolar state, osteoporosis, avascular necrosis of the hip, peptic/gastric ulcer disease, cataracts and glaucoma, steroid myopathy, cushingoid appearance, weight gain, suppression of the pituitary-hypothalamic axis, and growth suppression in children; water retention may precipitate congestive heart failure and hypertension; hypokalemia; unmasking of latent infections (eg, tuberculosis, herpes zoster) and predisposition to fungal and parasitic infection; due to suppressed pituitary-hypothalamic axis, additional steroid dosing may be necessary at time of stress (eg, systemic infections, surgery)

### Drug Category: *Immunosuppressives*

If the overlying condition is inflammatory or autoimmune in nature, immunosuppressives may be of limited benefit to those patients intolerant of steroids.

<b>Drug Name</b>	Intravenous immune globulin (IVIG)
<b>Description</b>	Neutralize circulating myelin antibodies through anti-idiotypic antibodies. Down regulates proinflammatory cytokines, including IFN-gamma. Blocks Fc receptors on macrophages. Suppresses inducer T cells and B cells and augments suppressor T cells. Blocks complement cascade, promotes remyelination, and may increase CSF IgG (10%).
<b>Adult Dose</b>	2 g/kg IV over 2-5 d
<b>Pediatric Dose</b>	Not established
<b>Contraindications</b>	Documented hypersensitivity; IgA deficiency
<b>Interactions</b>	Globulin preparation may interfere with immune response to live virus vaccine (MMR) and reduce efficacy (do not administer within 3 mo of vaccine)
<b>Pregnancy</b>	C - Safety for use during pregnancy has not been established.
<b>Precautions</b>	Check serum IgA before IVIG with IgA-depleted product (eg, Gammagard S/D); infusions may increase serum viscosity and thromboembolic events; infusions may increase risk of migraine attacks, aseptic meningitis (10%), urticaria, pruritus, or petechiae (2-5 d postinfusion to 30 d); increases risk of renal tubular necrosis in elderly patients and in patients with diabetes, volume depletion, and preexisting kidney disease; laboratory results with changes that are associated with infusions include elevated antiviral or antibacterial antibody titers for 1 mo, 6-fold increase in ESR for 2-3 wk, and apparent hyponatremia

### Drug Category: *Anticonvulsants*

To treat associated symptom of dysesthesia and neuropathic pain.

<b>Drug Name</b>	Gabapentin (Neurontin)
<b>Description</b>	Has anticonvulsant properties and antineuralgic effects; however, exact mechanism of action is unknown. Structurally related to GABA but does not interact with GABA receptors. Titration to effect can take place over several days (300 mg on day 1, 300 mg bid on day 2, and 300 mg tid on day 3).

<b>Adult Dose</b>	Day 1: 100 mg PO tid or 300 mg hs Day 2: 400 mg PO tid over 3 d and titrate prn; not to exceed 1200 mg PO qid
<b>Pediatric Dose</b>	<12 years: Not established >12 years: Administer as in adults
<b>Contraindications</b>	Documented hypersensitivity
<b>Interactions</b>	Antacids may significantly reduce bioavailability of gabapentin (administer at least 2 h following antacids); may increase norethindrone levels significantly
<b>Pregnancy</b>	C - Safety for use during pregnancy has not been established.
<b>Precautions</b>	Caution in severe renal disease

<b>Drug Name</b>	Carbamazepine (Tegretol)
<b>Description</b>	DOC that may reduce polysynaptic responses and block posttetanic potentiation.
<b>Adult Dose</b>	100 mg PO bid initially, increase by 200 mg/d with 100-mg increments q12h prn; not to exceed 1200 mg/d
<b>Pediatric Dose</b>	<12 years: Not established >12 years: Administer as in adults
<b>Contraindications</b>	Documented hypersensitivity; history of bone marrow depression; administration of MAO inhibitors within last 14 d
<b>Interactions</b>	Serum levels may increase significantly within 30 days of danazol coadministration (avoid whenever possible); do not coadminister with monoamine oxidase (MAO) inhibitors; cimetidine may increase toxicity, especially if taken in first 4 wk of therapy; may decrease primidone and phenobarbital levels (their coadministration may increase carbamazepine levels)
<b>Pregnancy</b>	D - Unsafe in pregnancy
<b>Precautions</b>	Do not use to relieve minor aches or pains; caution with increased intraocular pressure; obtain CBCs, LFTs, and serum-iron baseline prior to treatment, during first 2 months, and yearly or every other year thereafter; can cause drowsiness, dizziness, and blurred vision; caution while driving or performing other tasks requiring alertness

<b>Drug Name</b>	Zonisamide (Zonegran)
<b>Description</b>	Indicated for adjunctive treatment of partial seizures with or without secondary generalization. Evidence that zonisamide is effective in myoclonic and other generalized seizure types exists.
<b>Adult Dose</b>	100 mg/d PO for 2 wk, then increase by 100 mg/d PO q2wk to maximum of 400 mg/d; may be given qd or divided bid
<b>Pediatric Dose</b>	Not established
<b>Contraindications</b>	Documented hypersensitivity
<b>Interactions</b>	May increase serum carbamazepine levels; carbamazepine may increase zonisamide concentrations; phenobarbital may decrease zonisamide levels
<b>Pregnancy</b>	C - Safety for use during pregnancy has not been established.
<b>Precautions</b>	May cause drowsiness, weight loss, ataxia, nausea, and slowing of mental activity; recommended that patients drink at least 6-8 glasses of water daily to prevent kidney stones; do not use in patients with glomerular filtration rate <50 mL/min; caution in patients with renal and hepatic dysfunction

<b>Drug Name</b>	Topiramate (Topamax)
<b>Description</b>	Sulfamate-substituted monosaccharide with broad spectrum of antiepileptic activity that may have state-dependent sodium channel blocking action. Potentiates inhibitory activity of neurotransmitter GABA. In addition, may block glutamate activity. Not necessary to monitor plasma concentrations to optimize therapy. On occasion, addition of topiramate to phenytoin may require adjustment of phenytoin dose to achieve optimal clinical outcome.
<b>Adult Dose</b>	50 mg/d PO; titrate by 50 mg/d at 1-wk intervals to target dose of 200 mg bid; not to exceed 1600 mg/d
<b>Pediatric Dose</b>	<2 years: Not established 2-16 years: 1-3 mg/kg PO initially; not to exceed 25 mg/d, then titrate dose upward by 1-3 mg/kg/d divided bid (not to exceed dosage increases of 25 mg) q1-2wk until total daily dose is 5-9 mg/kg/d divided bid >16 years: Administer as in adults
<b>Contraindications</b>	Documented hypersensitivity
<b>Interactions</b>	Phenytoin, carbamazepine and valproic acid can significantly decrease levels; reduces digoxin and norethindrone levels when administered concomitantly; concomitant use with carbonic anhydrase inhibitors may increase risk of renal stone formation and should be avoided; use with extreme caution when administering concurrently with CNS depressants because may have an additive effect in CNS depression and in other cognitive or neuropsychiatric adverse events
<b>Pregnancy</b>	D - Unsafe in pregnancy
<b>Precautions</b>	Risk of developing a kidney stone is increased 2-4 times that of untreated population; risk may be reduced by increasing fluid intake; caution in renal or hepatic impairment Patients should seek immediate medical attention if they experience blurred vision or periorbital pain; continued usage after symptoms develop, can lead to glaucoma; primary treatment is discontinuation of topiramate; if left untreated, serious sequelae, including permanent vision loss, may occur Oligohidrosis and hyperthermia has been reported predominantly in children during vigorous exercise or exposure to warm environmental temperatures (ensure proper hydration prior to and during activity and warm temperatures) May cause hyperchloremic, nonanion gap metabolic acidosis with acute or chronic metabolic acidosis, resulting in hyperventilation and nonspecific symptoms such as fatigue and anorexia or more severe adverse effects including cardiac arrhythmias or stupor; chronic untreated metabolic acidosis may increase risk for nephrolithiasis or nephrocalcinosis risk, osteomalacia (ie, rickets in pediatric patients), or osteoporosis (with an increased risk for fractures); chronic metabolic acidosis in pediatric patients may also reduce growth rates; measure baseline and periodic serum bicarbonate

<b>Drug Name</b>	Pregabalin (Lyrica)
<b>Description</b>	Structural derivative of GABA. Mechanism of action unknown. Binds with high affinity to alpha2-delta site (a calcium channel subunit). In vitro, reduces calcium-dependent release of several neurotransmitters, possibly by modulating calcium channel function. FDA approved for neuropathic pain associated with diabetic peripheral neuropathy or postherpetic neuralgia and as adjunctive therapy in partial-onset seizures.
<b>Adult Dose</b>	50 mg PO tid initially; if needed, may increase to 100 mg tid within 1 wk

<b>Pediatric Dose</b>	Not established
<b>Contraindications</b>	Documented hypersensitivity
<b>Interactions</b>	May cause additive effects on cognitive and gross motor functioning when coadministered with drugs that cause dizziness or somnolence
<b>Pregnancy</b>	
<b>Precautions</b>	Discontinue gradually (over a minimum of 1 wk) to minimize increased seizure frequency in patients with seizure disorders; may cause insomnia, nausea, headache, or diarrhea with abrupt withdrawal; common adverse effects include dizziness, somnolence, blurred vision, weight gain, and peripheral edema; may elevate creatinine kinase level, decrease platelet count, and increase PR interval; doses >300 mg/d associated with higher rate of adverse effects and treatment discontinuation; decrease dose with renal impairment (ie, CrCl <60 mL/min)

<b>Drug Name</b>	Zonisamide (Zonegran)
<b>Description</b>	Indicated for adjunctive treatment of partial seizures with or without secondary generalization. Evidence suggests efficacy for myoclonic and other generalized seizure types.
<b>Adult Dose</b>	100 mg/d PO for 2 wk, then increase by 100 mg/d q2wk; not to exceed 400 mg/d; may be given qd or bid
<b>Pediatric Dose</b>	Not established
<b>Contraindications</b>	Documented hypersensitivity
<b>Interactions</b>	May increase serum carbamazepine levels; carbamazepine may increase concentrations; phenobarbital may decrease levels
<b>Pregnancy</b>	C - Safety for use during pregnancy has not been established.
<b>Precautions</b>	May cause drowsiness, weight loss, ataxia, nausea, and slowing of mental activity; pediatric patients have an increased risk for oligohidrosis and hyperthermia

### Drug Category: *Tricyclic antidepressants*

Analgesic for chronic and neuropathic pain.

<b>Drug Name</b>	Amitriptyline (Elavil)
<b>Description</b>	Inhibits reuptake of serotonin and/or norepinephrine at presynaptic neuronal membrane, which increases concentration in CNS. May increase or prolong neuronal activity since reuptake of these biogenic amines is important physiologically in terminating transmitting activity.
<b>Adult Dose</b>	10-300 mg/d PO hs
<b>Pediatric Dose</b>	Children: 0.1 mg/kg PO hs; increase, as tolerated, over 2-3 wk to 0.5-2 mg/d hs Adolescents: 10-50 mg/d initially; increase, as tolerated, gradually to a maximum of 300 mg/d in divided doses
<b>Contraindications</b>	Documented hypersensitivity; administration of MAOIs in past 14 d; history of seizures, cardiac arrhythmias, glaucoma, or urinary retention
<b>Interactions</b>	Phenobarbital may decrease effects; coadministration with Cyp2D6 enzyme system inhibitors (eg, cimetidine, quinidine) may increase levels; inhibits hypotensive effects of guanethidine; may interact with thyroid medications, alcohol, CNS depressants, barbiturates, and disulfiram

<b>Pregnancy</b>	D - Unsafe in pregnancy
<b>Precautions</b>	Caution in cardiac conduction disturbances, history of hyperthyroidism, and renal or hepatic impairment; avoid in patients who are elderly

## FOLLOW-UP

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### Further Inpatient Care

- Inpatient care should be directed as appropriate for the treatment of the primary disease.

### Further Outpatient Care

- The patient should follow up with the primary physician for underlying disorders (eg, diabetes).
- The patient should follow up with the primary physician or with the rehabilitation physician for pain medications and/or monitoring of laboratory tests.

### In/Out Patient Meds

- Over-the-counter analgesics are used for pain control.
- Prescription pain medications: The author of this article searched recent literature for treatment of neuropathic pain; the search returned 925 possible references, none of which advocated the use of opioid medication for neuropathic pain, either short term or long term.
- Various other medications may be used to reduce dysesthetic pain, which typically is a stabbing burning pain.
  - Traditional medical models for treating chronic and neuropathic pain are based on the use of tricyclic antidepressants (eg, amitriptyline, nortriptyline, desipramine). When appropriate, anticonvulsant drugs have been used to treat lancinating pain. Gabapentin, phenytoin, and carbamazepine most often are used as the drugs of choice. Valproate is used occasionally.
  - Advances in understanding of the pathophysiology of acute and chronic pain states may lead to newer, more effective pharmacologic approaches to treatment. The clinician must integrate symptoms, signs, and clinical evaluation when considering the treatment of neuropathic pain.
  - More effective and safer antiepileptic drugs have continued to benefit patients with conditions of chronic pain. Neurotransmitters such as serotonin, glutamate, substance P, CGRP, and GABA are the targets of research and development of pharmacologic therapies for acute and chronic pain. In addition, sodium activity and calcium activity play important roles in the pathology and treatment of these chronic medical problems.
  - Medications that increase gastric motility (eg, Reglan) may be administered. Gastric motility also may be increased by eating small frequent meals and by sleeping with the head elevated.
- Medications for bladder dysfunction – include bethanechol and oxybutynin.

### Complications

- Recurrent or unnoticed injury to any part of the body Deformity
- Deformity
- Atrophy
- Disturbances of organ functions that are autonomically controlled (eg, cardiac, gastric, bladder)
- Decreased self-esteem and decreased social interaction due to inability to participate in activities because of pain or incoordination
- Relationship problems associated with impotence

## Prognosis

- Recovery typically is the course, and it may take months to years. The same syndrome has a tendency to recur after an interval of months or years.

## Patient Education

- If the causative factor is discovered, education is directed toward avoidance of the initiating cause or pathogen. Additionally, recognition of early symptomology should be encouraged so that early treatment can be sought.
- Persons with one occurrence of mononeuritis multiplex are more prone to a recurrence.
- Persons with decreased sensation should be instructed to frequently check their feet or other affected areas for bruises, cuts, wounds, or other injuries. Also, patients who are insensate or incapacitated should be instructed to avoid prolonged pressure on various points on the body (eg, knees, elbows, sacrum) so as to avoid the development of pressure sores or ulcers.
- Safety awareness instruction is important to these patients because of their impaired sensation and decreased ability to compensate for limitations. The patient should be instructed to assure there is always adequate lighting, to test the water temperature before bathing or immersing body parts, and to wear protective shoes (no open toes or high heels).

### MISCELLANEOUS

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## Medical/Legal Pitfalls

- The main potential medicolegal pitfall stems from not obtaining a complete history and physical examination, thus allowing the main underlying disease process to go undetected/undiagnosed. As mentioned previously, not initiating early treatment for vasculitis could result in death. Legal consequences could arise should one not recognize an early viral infection or the symptomology of HIV, thus leading to delayed treatment.
- Additional medicolegal liability may arise from not fully informing patients of potential adverse side effects of medications.
- Both the physician and the patient should have the understanding that the nerve pain may be persistent for an

extended period and may require ongoing treatment, with possible referral to a comprehensive pain treatment center. Both must have realistic expectations.

## REFERENCES

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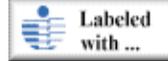
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